CASE REPORTS

Wheeze and *Mycoplasma pneumoniae*

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There is increasing interest in whether the atypical organisms *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* can cause exacerbations of asthma[1,2]. I describe four previously fit non-asthmatic patients who developed severe wheeze and airways obstruction in association with *M. pneumoniae* and who made a full recovery. When atypical asthma is associated with systemic symptoms the differential diagnosis should include *M. pneumoniae* infection.

CASE HISTORIES

The four patients were previously fit with no history of asthma and they presented with dyspnoea, chest tightness and malaise. All were wheezy with an obstructive pattern of spirometry (Table 1). Initial white blood cell counts were raised (range 12.6–16.8 $\times 10^9\,L^{-1}$; predominant neutrophilia). They were not anaemic and the initial erythrocyte sedimentation rate was high (range 93–125 mm/h). Chest radiographs (CXR) showed bilateral fine infiltrates. The diagnosis of *M. pneumoniae* infection was based on raised IgM antibodies and raised or rising titres of complement fixing antibodies against *M. pneumoniae* (range $>1{:}256$–$>1{:}512$). Symptoms, abnormal investigations and radiographs eventually resolved and no patient has subsequently needed treatment for wheeze or asthma.

Case 1

A man of 45 had a one-month history of cough and dyspnoea associated with sore throat, headache and malaise. He looked unwell with bi-basal crackles and wheeze. He was treated with inhaled corticosteroids and oral erythromycin. Over the next two months his wheeze and CXR changes resolved.

Case 2

A woman aged 59 was admitted with a two-week history of dyspnoea, wheeze, non-productive cough and sore throat. She had bilateral inspiratory crackles and loud expiratory 'squeaks' and was hypoxic (PaO_2 6.8 kPa). She was treated with intravenous (iv) cephradine and clarithromycin with prednisolone 30 mg/day. She was in hospital for ten days and her wheeze slowly improved. Two months after admission she had fully recovered.

Table 1 **Spirometry values at time of initial presentation to hospital and after recovery**

	PEFR (L/min)		FEV1 (L)		FVC (L)		FEV1/FVC (%)	
Patient	Initial	Final	Initial	Final	Initial	Final	Initial	Final
1	400	540	2.3	3.8	3.3	5.0	70	76
2	100	460	1.1	1.8	1.6	2.5	69	72
3	300	620	1.9	3.0	2.8	3.7	68	81
4	350	430	1.3	2.7	2.0	3.5	65	77

PEFR=peak expiratory flow rate; FEV1=forced expired volume in first second; FVC=forced vital capacity

Case 3

A man of 53 was admitted with a two-week history of dyspnoea, chest tightness, malaise and aches. He had tachycardia, tachypnoea and hypoxia (PaO_2 6.5 kPa). There were bilateral inspiratory crackles with a harsh expiratory wheeze. He was treated with iv cefuroxime and clarithryomycin plus oral prednisolone 40 mg/day. Because he was still unwell after a week, bronchoscopy was performed. Transbronchial biopsies showed normal alveoli and scanty inflammation in the bronchial mucosa. Eleven days after admission he rapidly improved and was discharged on a reducing dose of prednisolone. Three months later he was symptom-free and off all treatment.

Case 4

A 63-year-old man had a one-month history of wheeze and dyspnoea associated with dry cough, malaise and weight loss. On examination he had bi-basal crackles and expiratory wheeze. He was treated with oral erythromycin and prednisolone 30 mg/day. Prednisolone was gradually reduced and two months later he was symptom-free.

COMMENT

These four patients with wheeze, dyspnoea and airways obstruction proved to be infected with *M. pneumoniae*. Presentation was unusual for community-acquired pneumonia in that wheeze was a prominent feature, the onset was gradual with lack of fever, and the CXR appearances were atypical. *M. pneumoniae* has a predilection for the respiratory epithelium and a luminal infiltrate can affect bronchi, bronchioles and alveoli[3]. Sore throat and cough are common and the disease can 'march' from the upper to the lower airways[3].

In view of its ability to produce airway inflammation it would be understandable if *M. pneumoniae* infection were

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to exacerbate preexisting asthma. Gil *et al*.[4] cultured *M. pneumoniae* from throat swabs in 25% of asthmatics compared with 6% of controls. Airway colonization might increase chronic inflammation and make asthma more difficult to control. Other workers have looked for *M. pneumoniae* using the polymerase chain reaction in bronchial lavage specimens obtained at bronchoscopy. The organism was detected in 10 out of 18 patients with chronic asthma and in only 1 of 11 controls[5]. It is increasingly suggested that *C. pneumoniae* too can exacerbate asthma[1,2].

M. pneumoniae infection can cause wheeze in children[6] but it is not clear how commonly it causes wheeze in non-asthmatic adults. In a large study comparing features in adult pneumonia caused by different organisms wheeze was not assessed[7]. However, in keeping with the findings in my four patients, people with *M. pneumoniae* infection tended to be younger with a longer history and more frequent upper respiratory symptoms[7]. Although spirometric values were not reported, some authors have noted that in the acute phase of mycoplasma pneumonia a coexisting obstructive and restrictive picture is common and can be slow to resolve[8]. Another study found evidence of *M. pneumoniae* infection in only 3 out of 151 admissions for severe asthma[9]. Mycoplasma infections occur in cyclical epidemics and how commonly they are reported to produce wheeze may depend on whether or not it is an epidemic year. In adults, mycoplasma-associated bronchiolitis without pneumonia has occasionally been reported[10]. Asthma is probably uncommon in *M. pneumoniae* infection as a study of 15 adults showed no evidence of airways obstruction[11].

It is not possible to say whether resolution of wheeze in these cases was hastened by use of macrolide antibiotics and/or corticosteroids (oral in three, inhaled in one), but this is likely. Macrolides can have anti-inflammatory as well as antibiotic actions[12]. In light of the current interest in the relationship between *M. pneumoniae* infection and asthma[1,2], it is reassuring to note that severe wheeze caused by this organism can fully resolve.

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Refractory coeliac disease, small-bowel lymphoma and chorea

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Various neurological disorders have been described in coeliac disease[1,2]. They have not so far included chorea.

CASE HISTORY

At age 58 a woman was investigated for symptomatic anaemia and found to be iron and folate deficient. She had no abdominal symptoms. Findings on upper gastrointestinal endoscopy were normal but distal duodenal biopsies showed subtotal villous atrophy, inflammatory infiltration of the lamina propria and an increase in intraepithelial lymphocytes. Coeliac disease was diagnosed and she was started on a gluten-free diet. She gained weight and ceased to be anaemic; however, repeat biopsies of her duodenal mucosa showed no improvement in the villous atrophy. Compliance with a gluten-free diet was confirmed by dietitians' assessments. She subsequently became hypothyroid with a 1 in 25 600 antithyroid antibody titre, and was treated with thyroxine.

11 years later she complained of diarrhoea, ankle oedema and 7 kg weight loss. Endoscopic duodenal biopsies again showed features consistent with untreated coeliac

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disease, and again a dietitian confirmed adherence to a strict gluten-free diet. Her haemoglobin was 12.6 g/dL, mean corpuscular volume 82 fL, albumin 33 g/L (normal 35–50), antiendomysial antibody positive, vitamin B12 normal and red cell folate low at 145 ng/mL (normal > 185). A barium follow-through showed a rather featureless jejunal mucosa but no obstructive lesion of the small bowel; nothing abnormal was seen on an ultrasound scan of the abdomen. In view of her worsening symptoms despite a gluten-free diet, she was started on prednisolone, initially 30 mg daily, and over the next year her clinical condition improved, with resolution of her diarrhoea and a weight gain of 5 kg. At the time of discontinuation of prednisolone a year later she developed involuntary writhing movements of her left limbs. On examination she had choreo-athetoid movements involving the left limbs and persistent dysarthria. There was no associated weakness and sensation was normal. At that time she was taking digoxin, aspirin, ferrous sulphate and thyroxine; she had never received any neuroleptic medication. Magnetic resonance scanning of the brain and brainstem was normal, with no evidence of an infarct and no lesion in the basal ganglia. Genetic testing for Huntington's disease—a search for the hallmark abnormal CAG repeats on chromosome 4—was negative. No underlying neurological disease to explain her chorea was found.

Her chorea continued and seven months later she developed acute abdominal pain. At emergency laparotomy she was found to have a perforated jejunal tumour, which was resected. It proved to be a non-Hodgkin's T-cell lymphoma. She subsequently had intravenous chemotherapy with doxorubicin, cyclophosphamide and vincristine, but deteriorated three months postoperatively and died.

COMMENT

Apart from the lack of response to a gluten-free diet this patient had typical coeliac disease, and subsequently developed the recognized complication of T-cell lymphoma of the jejunum. There is an argument, however, that patients who do not respond histologically to a gluten-free diet do not strictly have coeliac disease. Enteropathy-associated T-cell lymphoma is most likely to develop in coeliac patients who either do not adhere to[3] or do not respond to[4,5] a gluten-free diet. The prognosis for this tumour is poor.

Various different neurological abnormalities have been recognized in coeliac disease[1,2]. The most common are ataxia, neuropathy and epilepsy. We have found no previous report of chorea. Our patient had no associated neurological disease, no history of taking drugs with extrapyramidal effects and no genetic markers for Huntington's chorea. Although her chorea may have been a neurological association of her refractory coeliac disease, an alternative explanation is that it was a paraneoplastic complication of her impending T-cell lymphoma. Chorea has been described as a paraneoplastic phenomenon in patients with non-Hodgkin's lymphoma[6], but to our knowledge this is the first case where it has been associated with lymphoma arising from a background of coeliac disease. The finding of chorea in association with coeliac disease should prompt a search for possible underlying intestinal T-cell lymphoma.

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Bilateral knee pain with hyperparathyroidism

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Brown tumours of hyperparathyroidism are seldom seen in the lower limb.

CASE HISTORY

A man of 56 was seen at the orthopaedic clinic with a 3–4 year history of bilateral knee pain. This was worsening, and the knee had become swollen. On examination there was retropatellar tenderness and the patello-femoral joint was painful on movement. Abnormal bony architecture was

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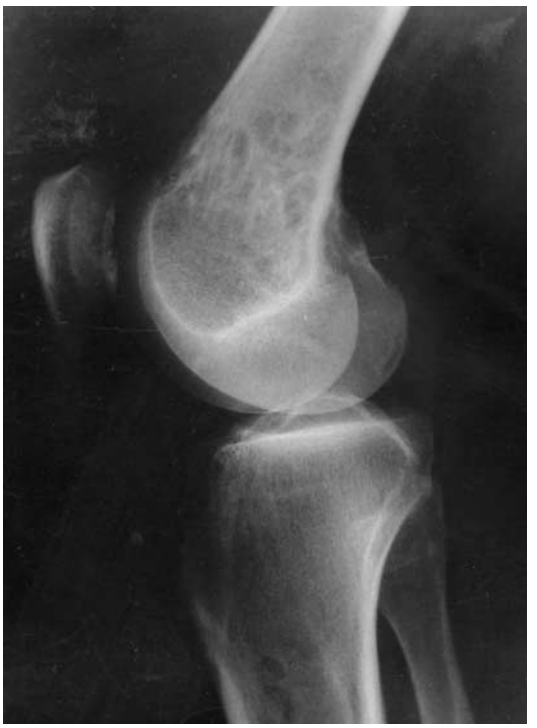

Figure 1 **Plain radiograph of knee joint**

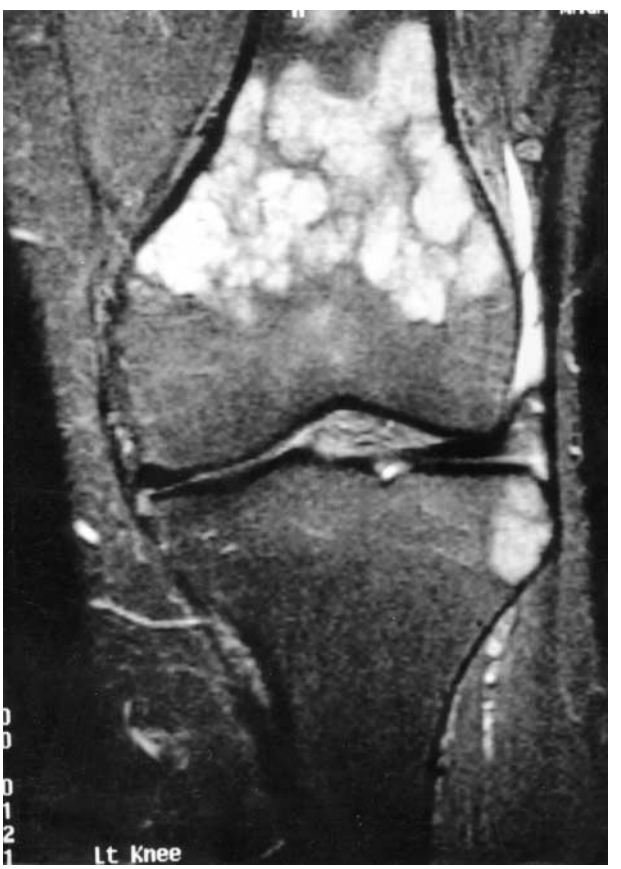

Figure 2 **MRI scan of knee**

seen on plain radiography (Figure 1). While awaiting an MRI scan he received physiotherapy, which exacerbated the pain. The MRI scan (Figure 2) showed multiple bone lesions, lobulated masses of low signal, in the lower femora. There was also breakthrough of the cortex. The menisci and cruciates were normal. The changes were consistent with a lymphatic or vascular abnormality, but all the blood tests (including myeloma screen and bone profile) were normal apart from a high serum calcium, 3.42 mmol/L. Vitamin D metabolites and 24-hour urinary calcium were not measured. The patient had noticed a swelling on the right side of his mandible and was referred to the oral and maxillofacial team. On plain radiography there was a radiolucency between the two premolar teeth, and biopsy showed a brown tumour of hyperparathyroidism. The parathyroid hormone concentration was 553 mmol/L (normal range 10–70). On ultrasonography there was no evidence of a mass lesion in the neck. A skeletal survey was not performed.

Neck exploration, undertaken by the general surgical team, revealed a 2 cm diameter left parathyroid adenoma, which was completely excised; a normal parathyroid gland was also excised. Postoperatively the patient became hypocalcaemic, and needed intravenous calcium. Oral calcium and vitamin D supplements were also prescribed. At follow-up serum calcium had stabilized at 2.20 mmol/L, alkaline phosphatase was normal, the bony symptoms had completely resolved, and plain radiographs of the femora showed resolution of the previous abnormalities.

COMMENT

Brown tumours of bone or osteitis fibrosa cystica (von Recklinghausen's disease of bone) are a feature of longstanding hyperparathyroidism. Microscopically, there is a combination of osteoblastic and osteoclastic activity, with associated cyst formation. Giant cells, haemosiderin-laden macrophages and fibroblasts fill the lytic lesions. These are not neoplasms and the brown appearance results from vascularity, haemorrhage and haemosiderin. They also contain fibrous tissue.

The earliest bony changes in hyperparathyroidism are seen in radiographs of the hands, where subperiosteal erosions can be detected in the phalanges, especially on the radial aspect of the middle phalanx and terminal tufts. The skull acquires a mottled appearance with lucent cystic areas ('pepperpot skull').

In retrospect, the lesion in our patient's knee was probably a brown tumour, though a biopsy was not done. Brown tumours are now rare in the UK because biochemical assays allow early diagnosis of hyperparathyroidism. The usual sites of these lesions are the ribs, clavicles, pelvic girdle and facial bones[1] and the presentation

may be a pathological fracture. The lower limb is seldom affected. Lloyd[2] describes the case of a 14-year-old boy with genu valgum, apathy, chorea, ataxia and hypercalcaemia. Osteitis fibrosa cystica was seen in the metaphyses of both femora, and iliac crest biopsy showed brown tumour.

In Asia the condition is more commonly encountered. From India, Mishra *et al.*[3] report 20 patients with primary hyperparathyroidism and brown tumours in the spine, pelvis and lower limbs, some associated with spontaneous fractures. These patients had a low mean age (38 years) and showed vitamin D deficiency with low bone mineral density. The authors postulate that, in these Indian patients, deficiency of vitamin D contributed to the pathogenesis of primary hyperparathyroidism.

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Perineal necrotizing fasciitis with dilatation of Cowper's gland

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In an immunocompromised patient, a urethral stricture can have life-threatening consequences.

CASE HISTORY

A man aged 35 was worried by a painless lump below his scrotum, present for some 6 months. He also complained of obstructive voiding symptoms but denied any history of trauma or urethritis. He had been diagnosed with HIV four years previously and was receiving triple therapy with a CD4 count of 150×10^9/L. On examination he had a lump in his perineum the size of a golf ball. An ultrasound scan suggested a urethral diverticulum. He did not attend for follow-up as requested but returned 5 months later with a large fluctuant perineal mass, with ischaemic changes to the overlying skin (Figure 1). An ultrasound and urethrogram showed a tight bulbar stricture with a track via Cowper's duct and gland into the perineum (Figure 2). Later that day he underwent urethroscopy, optical urethrotomy and debridement of necrotizing fasciitis. A large infected urinoma was drained and the track to Cowper's gland was curettaged. 4 months later he was well, with a normal urethrogram.

COMMENT

Cystic dilatation of the main duct of Cowper's bulbo-urethral gland is well documented[1,2]. This case, however, demonstrates not only dilatation of the duct and gland but also the development of a communication to the perineum and subsequent urinoma formation, seemingly not reported previously. A combination of a tight urethral stricture and the immunocompromised state of the patient led to the development of a life-threatening condition.

In his original description of infective necrotizing fasciitis Jean Alfred Fournier described a rapidly progressive genital gangrene that was idiopathic in origin[3]. This case reflects the emergence of HIV as a major risk factor in the development of the condition.

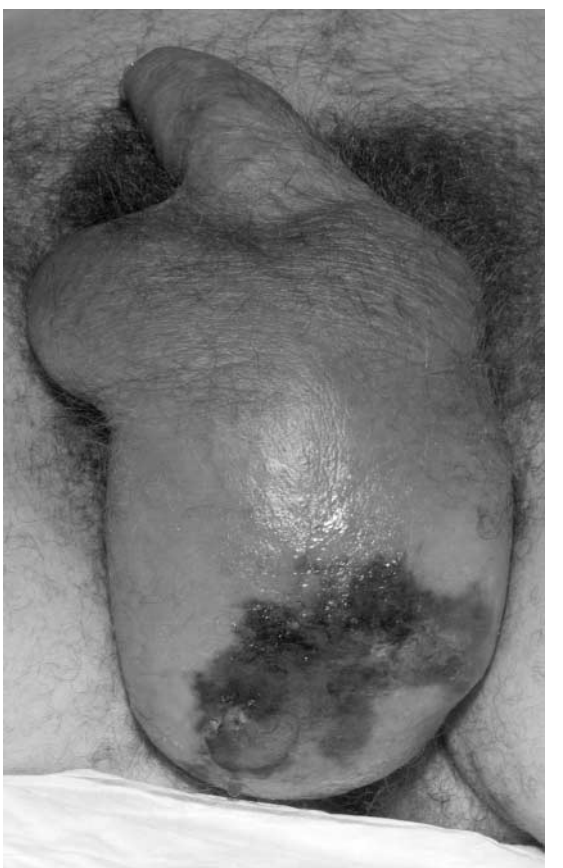

Figure 1 **Perineal necrotizing fasciitis**

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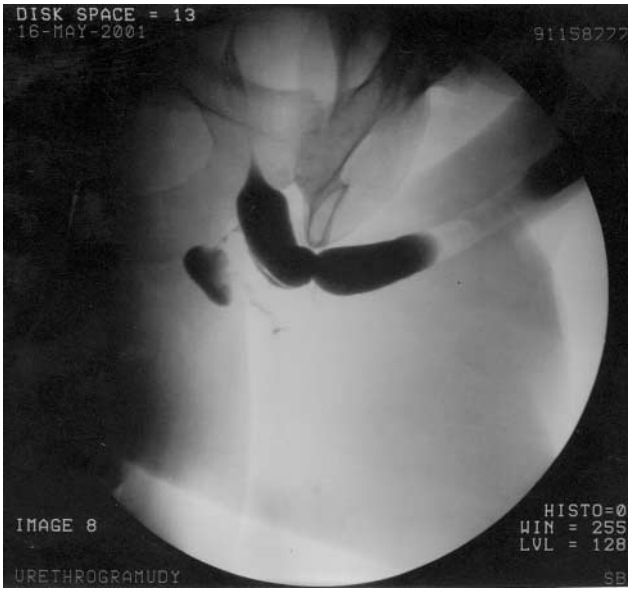

Figure 2 **Preoperative urethrogram**

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The man who walks backwards

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Backward walking is sometimes a manifestation of psychosis, but not always.

CASE HISTORY

A man aged 53 experienced trouble with coordination at the age of 5. He had had a difficult delivery. Nocturnal grand mal seizures developed at age 8, and between 18 and 21 severe myoclonus confined him to bed, surrounded by pillows in a high-sided cot because of uncontrollable flailing arm movements. He recovered apart from residual torticollis but 5 years later developed a depressive illness. Psychotic features emerged subsequently—persecutory delusions and episodes of bizarre behaviour accompanying a variety of changes in mood. He used a meat cleaver to prune shrubs and he was found arranging the branches across a road near the family home. Other unusual behaviours included sitting in a deck chair in the pouring rain. There was a partial relapse of his myoclonus at age 31.

In his early 40s the torticollis began to worsen and was accompanied by increasing lumbar spine pain as he twisted his torso to compensate for a deviated field of vision. An occupational therapist suggested he try walking backwards, and this he did with some success. Although finding road crossing difficult he became rapidly able to manage his shopping, pulling a trolley behind him. Friends nicknamed him 'The Sidewinder'. He exhibits a broad-based gait but no other cerebellar signs or focal neurological features. He now never walks forwards unless asked. Psychiatrically he has been symptom free for many years on a combination of lithium carbonate and antipsychotics. There is no evidence to suggest the backward walking is associated with any psychotic symptoms or disturbance of mood.

COMMENT

Although this patient had experienced psychotic illness, his backward walking is a functional accommodation to a physical disorder. It was clearly an act of will: to become automatic, backward walking requires a drastic change in the locomotor programme[1]. The patient is not embarrassed by his potentially stigmatizing behaviour. Neurologically he meets broad diagnostic criteria for the Ramsay–Hunt syndrome[2]—ataxia, myoclonus, grand mal—though no longer experiencing seizures. Intracranial disease is sometimes linked with mood disorders[3,4] such as he experienced but his CT scans, and electroencephalograms performed in adult life, have been normal.

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Cholecystitis after cholecystectomy

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When symptoms recur after cholecystectomy, the possibility of an inflamed remnant must be considered.

CASE HISTORY

A woman aged 81 sought advice because of right upper quadrant pain and belching. She had experienced similar symptoms 24 months previously, at which time investigations at a district general hospital had revealed gallstones and she had undergone urgent subtotal cholecystectomy. The original operation note records that dissection of Calot's triangle was difficult because of inflammation, therefore a cuff of gallbladder was left *in situ* and a partial cholecystectomy was performed without intraoperative cholangiography. The patient recovered without incident and was well until her symptoms recurred. She was tender in the right upper quadrant but blood tests, including enzymatic liver function tests, were all normal. An endoscopic retrograde cholangiopancreaticogram (ERCP) revealed a stone in a large gallbladder remnant (Figure 1). The bile duct was normal.

She was then referred to our unit and after detailed risk assessment she was listed for completion cholecystectomy. At operation there was a sizeable gallbladder remnant. Calot's triangle was dissected and cholecystectomy was completed. An intraoperative cholangiogram was normal. The operative specimen contained a solitary large stone which appeared to be a primary gallstone, and chronic cholecystitis was confirmed histologically. At review two months after surgery she had recovered well.

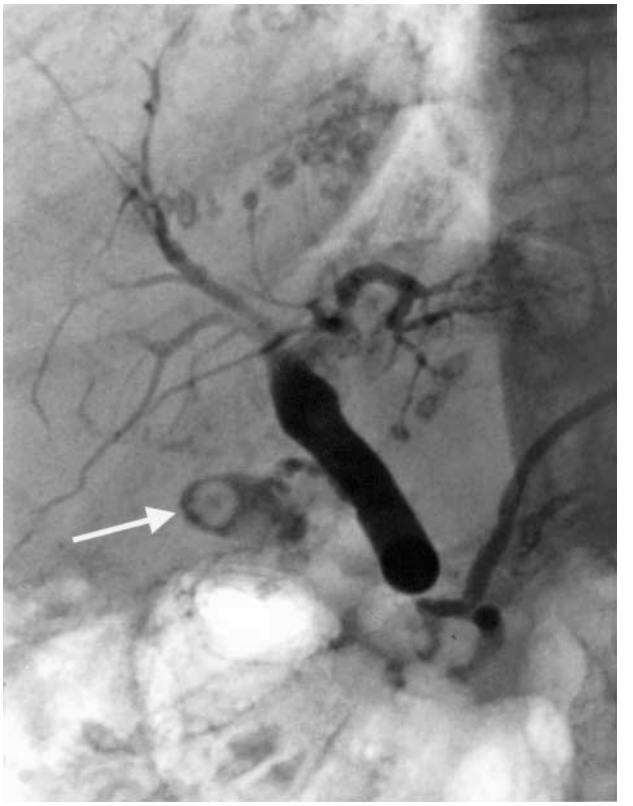

Figure 1 **Endoscopic retrograde cholangiopancreaticogram**

COMMENT

Partial cholecystectomy (leaving a cuff of gallbladder *in situ*) can be a good option in the context of acute inflammation since a potentially hazardous dissection of Calot's triangle is avoided[1]. However, it is important to ensure that the remnant of gallbladder that is left is free of stones and also that this portion of gallbladder is small.

In the case described here, the differential diagnosis at the time of re-presentation included bile duct stones and the post-cholecystectomy syndrome. Studies of post-cholecystectomy patients record continued symptoms in up to 50% of patients[2,3]. After assay of liver function tests and transabdominal ultrasonography, ERCP is probably the definitive test.

Recurrence of symptoms similar to those of cholecystitis after cholecystectomy should not be attributed to entities such as the post-cholecystectomy syndrome until there has been thorough disease reappraisal. Unless the clinician is certain that the cholecystectomy was complete, the possibility of recurrent inflammation in a remnant gallbladder must be considered. The only certain way to prevent recurrence of gallstones in the gallbladder is total cholecystectomy. Finally, the value of a careful history is emphasized by the patient's insistence that the symptoms at the time of recurrence were identical to those at the time of her original illness.

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The old lady who lived under a bridge

There was an old lady, lived under a bridge. She lived in a box, the box from a fridge.
Box from a fridge, under a bridge,
Poor old lady, I hope she lives.

She traded her insulin needles for cash, she dug through dumpsters, and ate from the trash.
Needles for cash, ate from the trash, box from a fridge, under a bridge,
Poor old lady, I hope she lives.

She was found unresponsive, someone called 911, her fingerstick glucose 901.
911, 901, needles for cash, ate from the trash, box from a fridge, under a bridge,
Poor old lady, I hope she lives.

They insulin dripped her and steriley draped her, then typed her and tapped her and tubed her and taped her.
Dripped her and draped her, tubed her and taped her, 911, 901, needles for cash, ate from the trash, box from a fridge, under a bridge,
Poor old lady, I hope she lives.

A month in the unit, they scanned her and scoped her, dozens of students all prodded and poked her,
Scanned her and scoped her, prodded and poked her, dripped her and draped her, tubed her and taped her, 911, 901, needles for cash, ate from the trash, box from a fridge, under a bridge,
Poor old lady, I hope she lives.

Finally stable, looked for nursing home beds, but she left AMA and she left without meds,
Nursing home beds, left without meds, scanned her and scoped her, prodded and poked her, dripped her and draped her, tubed her and taped her, 911, 901, needles for cash, ate from the trash, box from a fridge, under a bridge,
Poor old lady, I doubt she lives.

Eleven months later on a night that was freezing, severe hypothermia patient who's seizing
Went down to see this most interesting case, took a look at the chart, then remembered her face.

There was an old lady lived under a bridge
Poor old lady I hope she lives.

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